August 8, 2006

Writer's Direct Number: (317) 236-2120
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IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Re:	Invention:	METHOD OF TREATMENT FOR CENTRAL NERVOUS SYSTEM INJURY
	Inventors:	Richard B. Borgens; Scott A. Shapiro
	Serial No.:	10/748,572
	Filed:	December 30, 2003
	Art Unit:	1623
	Examiner:	Eric Olson
	Our Docket No.:	P01254-US-01 (19232.0011)

DECLARATION PURUSANT TO RULE 132

Richard B. Borgens, PhD., Declares and states:

1. I have served as the Director of the Purdue University Center for Paralysis Research, 408 South University Street, West Lafayette, IN 47907 since 1987. The Center for Paralysis Research is dedicated to research and testing dedicated to treating spinal cord injuries. As such, the majority of my professional career has been devoted to the meaningful pursuit of treatment for individuals suffering from spinal cord injuries.

2. I am also familiar with transdifferentiation as described in PCT Application WO01/08691 to Baranowitz et al. (the "Baranowitz Reference"), and I am the first named inventor for U.S. Patent No. 4,919,140 to Borgens et al. (the "Borgens Patent"). It is my understanding that claims 1 and 13 of the above-mentioned application has been rejected over the Baranowitz Reference in light of the Borgens Patent on the basis that it would have been

obvious to one of ordinary skill in the art at the time the invention was made to combine the use of transdifferentiating cells (creation of neurons from endothelial cells) with oscillating field stimulation as described in the Borgens Patent. I disagree with this analysis for the following reasons.

3. Inserting additional neurons does not restore nerve function. The spinal cord, like the brain, is composed of two sub-compartments: Gray and white matter. Gray matter is made up of several cell types, chiefly neurons (cell body or soma, containing the nucleus). In contrast, White Matter is composed of axons and these other supporting cells–there are no neurons in white matter. See Fig. 1 attached.

4. Most clinical spinal cord injuries are less than one vertebral segment in longitudinal extent[1,2]–more recent MRI measurements suggest this distance to be on the order of ~ 30mm (3). Spinal cord tissue dies after insult from the inside out (Central Hemorrhagic Necrosis) causing most if not all gray matter to be destroyed, as well as a significant part of the white matter over this relatively short distance of ~ 30 mm in longitudinal extent.[3,4]

5. It is well known in the art that the loss of the white matter component of the spinal cord that produces catastrophic functional loss, not the loss of gray matter and the neurons contained within. *See, e.g.* citations 5, 6 (emphasizing that spinal cord injury is a "white matter injury"). In a recent text "Restoring Function to the Injured Human Spinal Cord " (Springer - Verlag, 2003; citation 7), I am quoted as summarizing this fact:

> ... spinal cord injury resulting in quadriplegia or paraplegia is a white matter injury. It is the interruption of the long tract communication system between the body and brain that segments or compartmentalizes the injure body into two regions: functional and non – functional." (Page 7, chapter 2.1)

In fact when all of the gray matter, and the neurons within it is destroyed for 1 vertebral segment, but with variable levels of intact functional white matter – the result is Central Cord Syndrome[8,9]. The main symptoms of this are: (1) muscle weakness (paresis) and not paralysis as after severe spinal cord injury; and (2) a weakening of reflex tone (hyporeflexia) and not hyperreflexia—indicative of paralyzing spinal cord injury.

6. Therefore, even if transdifferentiation were used to replace the destroyed neurons (grey matter) in a spinal cord after a spinal cord injury, no meaningful regeneration of the white matter would be expected. To my knowledge, there is currently no evidence or reason to believe that use of OFS with such transdifferentiated cells would result in restoration of nerve function in an injured spinal cord. For this reason, transdifferentiation as discussed in the Baranowitz Reference is nonanalogous to the goal or function of any of the claims of the above-mentioned application, and does not result in growth of axons or dendrites on existing uninjured tissue.

7. Further, I am familiar with the research papers forming the basis for U.S. Patent No. 6,551,612 to Benowitz et al. (the "Benowitz Patent"), and I am the first named inventor for U.S. Patent No. 4,919,140 to Borgens et al. (the "Borgens Patent"). It is my understanding that claims 1 and 13 of the above-mentioned application has been rejected over the Benowitz Patent and Borgens Patent on the basis that it would have been obvious to one of ordinary skill in the art at the time the invention was made to combine the use of inosine with the use of oscillating field stimulation because the two prior applications would presumably show additive beneficial effects when combined. I disagree with this analysis because our understanding of the cited

literature and state of the art at the time of invention was as follows:

8. Delay after Injury renders Oscillating Field Stimulation ("OFS") impotent. OFS has been shown to be unable to produce useful functional change in naturally injured paraplegic dogs when treatment is delayed for more than 3 weeks after the original injury.[9,10] In addition, the behavioral outcome in both experimental and matched control animals in guinea pig testing indicated a failure to produce regeneration of neural function when treatment was delayed–even though over one hundred attempts were made to induce axonal regeneration or functional recovery in spinal injured guinea pigs with a delayed application. All of these attempts failed. Approximately 160 dogs were tested with delayed application of OFS, though none of them improved in a way that could be attributed to the OFS therapy (9,10).

9. Delay of over 100 hours post injury renders inosine application impotent. According to my understanding of the published literature and presentations from Benowitz et al. in 1999, and consistent with the Benowitz Patent, the efficacy of inosine in regenerating central nervous system function was limited to applications made within the first 100 hours of injury.[11]

10. Performing the treatment comprising a method covered by claim 1 resulted in unexpected and synergistic results—the treatment comprising a method covered by claim 1, when compared to subcutaneous inosine alone and a control group produced a statistically significant enhancement in the rate of functional recovery compared to inosine alone or the control. See results submitted as Figs.2 and 3 attached. OFS alone was not tested, as extensive prior testing showed OFS to be impotent under these circumstances.

11. In a treatment comprising a method covered by claim 1, all but one of the

recovering animals showed a CTM recovery by one month after the experimental application. By comparison, the recovery of the CTM was significantly delayed in response to the inosine only. This difference was statistically significant (P = 0.04; Fisher's Exact test, two-tailed) at a time post injury when the cited references indicate that neither treatment should be effective.

12. The treatment comprising a method covered by claim 1 produced a regeneration of long tract white matter axons after initial dieback of the cut fibers that is more robust than the treatment using inosine alone.

13. In ascending, largely sensory axon projections, significantly greater numbers of subjects treated according to the treatment comprising a method covered by claim 1 demonstrated axons regenerating across the plane of the transection into the adjacent segment of spinal cord than in the inosine alone subjects (6 of 9 vs. 2 of 12, respectively; P = 0.03; Table 1 attached). In Descending Tracts (largely motor axons), similar evidence of a significantly robust regeneration in response to the treatment comprising a method covered by claim 1 compared to subjects treated with inosine alone after blinded scoring of the termination of regenerating axons.

14. The subjects treated according to the treatment comprising a method covered by claim 1 was the only group showing evidence of a statistically greater termination of axons in all three zones close to the lesion: within 250 m of the plane of transection (P = 0.004), at the plane of the transection (P – 0.005), and crossing the lesion into the adjacent segment of spinal cord (P=0.04). Regenerating axons that had made up the distance after "dieback" to end at the level of the original plane of transection were statistically greater in number after the combination treatment when compared to the inosine alone therapy (P = 0.02, Table 2). All comparisons

were made with a conservative two-tailed statistical test that does not assume any standard distribution (non-parametric).

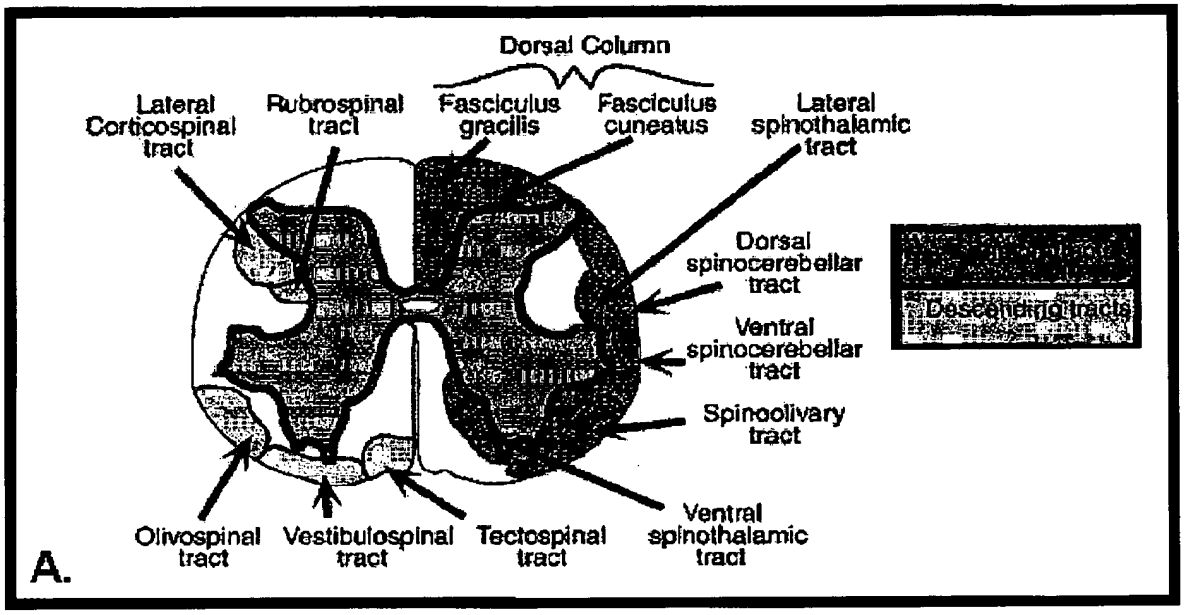
Under penalty of perjury, I declare that all statements made in this Declaration of my own knowledge are true and that all statements made on information and belief are believed to be true.

Date

Dr.Richard B. Borgens, Ph.D.

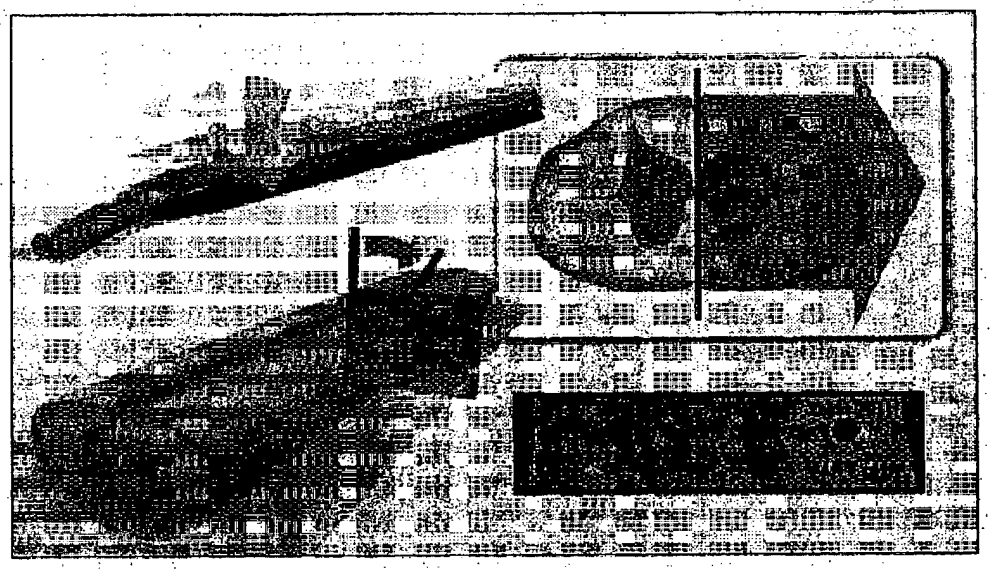
Citations

1. Bunge, R.P., Puckett. W.R., Becarra, J.L., et al., 1993. Observations of the Pathology of Human Spinal Cord Injury: A Review of 22 new cases with details from a case of chronic cord compression with extensive focal demyelination". Adv. in Neurolog. 59, 75 –89.
2. Tuswzynski,M.H., Gabriel, K., Gerhardt, K. and Szollar, S., 1999. Human spinal cord retains Substantial Structural Mass in chronic stages after injury. J. Neurotrauma, 16. 523 – 531
3. Metz GAS, Curt A, van de Meent H, Klusman I, Schwab ME, Dietz , 2000. Validation of the weight-drop contusion model in rats: A comparative study of human spinal cord injury .J. Neurotraauma 17): 1-17.
4. Yoganandan, L.A., Halliday,A.,Dickman,C,and E.benzel.1999. Practical Anatomy and fundamental Biomechanics in spine Surgery in Benzel edt., Spine Surgery, Curchill-Livingston, NY., 93 –0 118.
5. Blight, A.R.,1993. Remyelination, revascularization, and recovery of function in the experimental spinal cord injury. Advance in Neurobiology :Neural Injury, and Regeneration, 59, 91 – 103.
6. REIER PJ, STENSAAS LJ, GUTH L. (1983). The astrocytic scar as an impediment to regeneration in the central nervous system. In: Kao CC, Bunge R, Reier P, editors. Spinal Cord Reconstruction, New York: Raven Press p. 163-195.
7. Borgens,R.B., 2003. Restoring Function to the Injured Human Spinal Cord. Springer – Verlag, Heidleberg.
8. Borgens RB, Metacalf ME, Blight AR. (1993a). Delayed application of direct current electric fields in experimental spinal cord injuries. Restor Neurol Neurosci 5:173-179.
9. Borgens, RB, Toombs, JP, Blight, AR, McGinnis, ME, Bauer MS, et al., (1993b). Effects of applied electric fields on clinical cases of complete paraplegia in dogs. Restor Neurol Neurosci 5:305-322.
10. Borgens RB, Toombs JP, Breuer G, Widmer, WR, Waters, et al. 1999. An imposed oscillating electrical field improves the recovery of function in neurologically complete paraplegic dogs. J Neurotrauma 16:639-657.
11. Benowitz LI, Goldberg, DE, Madsen, JR, Sonid,I, N. 1999. Inosine stimulates extensive axon collateral growth in the rat corticospinal tract after injury. Proc Natl Acad Sci USA 96:13486-13490



A typical Cross Section of the Human Spinal Cord is shown; the Gray Matter (gray stippling) in the center contains neurons, glial support cells, and other cells and processes ,while the White Matter (outside this region) does not contain neurons. The White Matter is comprised of long tracts of nerve fibers (axons) that run largely parallel with the long axis of the cord, connecting body and brain. It is the interruption in this white matter that produces the catastrophic functional loss after SCI.

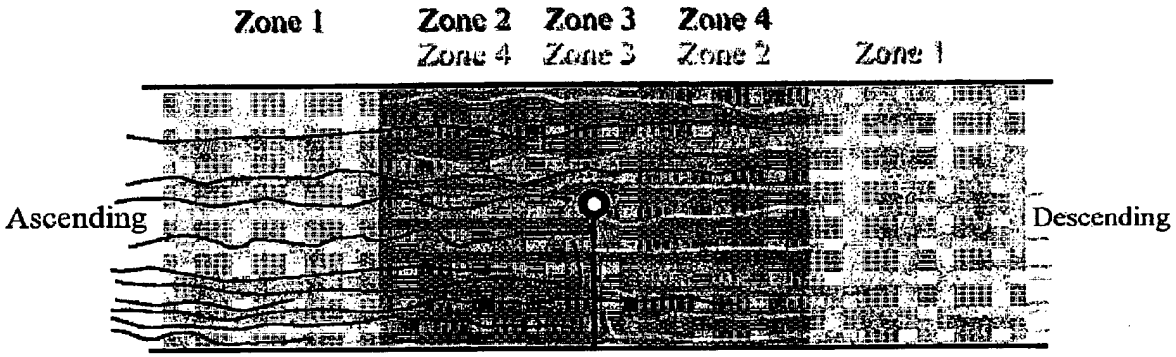
Fig 1



The drawing shows a hemisection (from the midline to the right margin of the cord) produced with a fine cutting instrument, and to its right, the plane of the transection (in rose). At the lower left, the placement of a platinum pin used to mark the plane of the right lateral hemisection is shown. Note that the marker is placed into the transection at the midline, and remains there held in place by scar tissue formation. Occasionally the marker shifts obliquely to the right as drawn. In the latter case this shifts the position of the marker hole away from midline as shown in the photomicrograph at the lower right (the marker is removed after fixation and before sectioning - see Methods). The plane of histological sectioning (in gray) is also shown in the cord at the bottom left. This procedure exactly marks the plane of transection(hatched line).

Fig. 2

Commissioner for Patents
Page 10



Ascending (Red)

	N	LTH	Zone 1 >250	Zone 2 <250	Zone 3	Zone 4
1. Control	15	5	10	3/10	0/10	0/10
2. Inosine	15	3	12	10/12	7/12	2/12
3. Inosine/ OFS	16	7	9	7/9	8/9	6/9
Statistics						
1 vs 2		0.68		0.03	0.005	0.48
1 vs 3		0.71		0.07	0.0001	0.003
2 vs 3		0.23		1.0	0.18	0.03

Descending (Yellow)

	N	LTH	Zone 1 >250	Zone 2 <250	Zone 3	Zone 4
1. Control	15	6	9	2/9	1/9	0/9
2. Inosine	15	7	8	7/8	2/8	1/8
3. Inosine/ OFS	16	5	11	10/11	9/11	5/11
Statistics						
1 vs 2		1.0		0.02	0.58	0.47
1 vs 3		0.7		0.004	0.005	0.04
2 vs 3		0.47		1.0	0.02	0.17

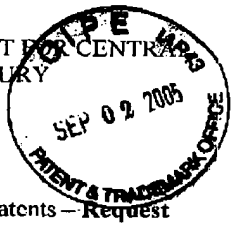
Fig. 3

Legend to Fig. 3

Ascending and Descending Axonal Projections after Experimental Applications
The drawing at the top diagrams the spinal cord – the head (rostral) to the right of the page, the tail (caudal) to the left. Note the position of the right lateral hemisection (severing only the right side of the spinal cord) as a heavy black line from the midline to the right margin of the drawing. Note also that anterogradely filled fibers diagrammed in yellow and red (filled from the caudal side and rostral side, respectively) project well past the plane of transection in undamaged white matter.
Note that on the right side of the cord, diagrammed fibers can terminate far short of the plane of transection (<250μm; zone 1 in dark gray), or project to within 250μm or less (zone 2) from the transection. Fibers were also observed terminating at the plane of transection, sometimes coursing along at its margin for short distances (zone 3), or they were observed to project into the adjacent segment of cord by usually passing around or through the transection plane (zone 4).
The Table provides the numbers of spinal cords (N) that were injected with the intracellular label and those that were lost to histology for each of the three groups. The proportions of those cords in which marked fibers were traced to the four zones are given over the number of cords examined. Statistical comparison between the groups is provided at the bottom of the graph (Fisher's Exact test, two-tailed). This data is given for both rhodamine labeled ascending fibers (in red) and fluorescein isothiocyanate (FITC) labeled descending projections (in yellow). Note that the number of cords lost to histology was not significantly different between any of the groups. An asterisk marks those comparisons that were statistically significantly different.

Mailed: August 30, 2005 via U.S. First Class Mail

Re: U.S. Patent Application
Serial No.: 10/748,572
Filed: December 30, 2003
Title: METHOD OF TREATMENT FOR CENTRAL NERVOUS SYSTEM INJURY
Inventor: Richard B. Borgens
Art Unit: 1614
Our File No.: P012544US-01 (19232.0011)



XX Transmittal letter to Commissioner for Patents – **Request for Correction of Inventorship**
XX Statement from Scott A. Shapiro
XX Declaration signed by Borgens and Shapiro (in two parts)
XX Check in the amount of $130.00
XX Assent of Assignee to Correction and/or Addition to Originally Named Inventors (signed by Purdue Research Foundation)
XX Return Postcard addressed to Thomas A. Walsh, Ice Miller

ICEMILLER
LEGAL & BUSINESS ADVISORS

WRITER'S DIRECT NUMBER: (317) 236-5946
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INTERNET: THOMAS.WALSH@ICEMILLER.COM

August 30, 2005

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

I hereby certify that this paper or fee is being deposited with the United States Postal Service as First Class mail in an envelope addressed to the Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450.

August 30, 2005
Date of Deposit

M. Kim Richardson
Printed or Typed Name of the Person Signing the Certificate

M. Kim Richardson
Signature

August 30 2005
Date of Signature

Re:	First Named Inventor:	BORGENS, Richard B.
	Invention:	METHOD OF TREATMENT FOR CENTRAL NERVOUS SYSTEM INJURY
	Serial No.:	10/748,572
	Filed:	December 30, 2003
	Our File No.:	P01254-US-01 (19232.0011)

REQUEST FOR CORRECTION OF INVENTORSHIP

Dear Sir/Madam:

Applicants request correction of inventorship to add Scott A. Shapiro as an inventor. The correct inventorship should be Richard B. Borgens and Scott A. Shapiro. Pursuant to 37 C.F.R. § 1.48 (1), this request is accompanied by:

(a) A statement from Scott A. Shapiro indicating that the error in inventorship occurred without deceptive intent on his part,

(b) A declaration signed by Richard B. Borgens and Scott A. Shapiro (in two parts),

(c) A check in the amount of $130,

(d) Written consent of the assignee, Purdue Research Foundation, and

(e) Return postcard.

In the event Applicants have inadvertently overlooked the need for payment of any additional fees, Applicants conditionally petition therefor, and authorize any deficiency to be charged to deposit account 09-0007. In the event the deposit account needs to be charged, it is requested that the number P01254-US-01 (19232.0011) be referenced.

If you have any questions regarding this correspondence, please feel free to contact the undersigned.

Respectfully submitted,

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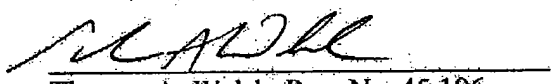

Thomas A. Walsh, Reg. No. 45,196
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TAW:mkr
Enclosures

PTO/SB/01 (03-01)(Amended by Customer pursuant to MPEP § 601.02)
Non-Amended Version Approved for use through 10/31/2002. OMB 0651-0032
U.S. Patent and Trademark Office; U.S. DEPARTMENT OF COMMERCE

DECLARATION AND POWER OF ATTORNEY FOR UTILITY OR DESIGN PATENT APPLICATION (37 CFR 1.63)

☐ Declaration Submitted with Initial Filing OR ☒ Declaration Submitted after Initial Filing (surcharge (37 CFR 1.16(e)) required)

Attorney Docket Number	P001254-US-01
First Named Inventor	Borgens
COMPLETE IF KNOWN	
Application Number	10/748,572
Filing Date	December 30, 2003
Group Art Unit	
Examiner Name	

As a below named inventor, I hereby declare that:

My residence, mailing address, and citizenship are as stated below next to my name.

I believe I am the original, first and sole inventor (if only one name is listed below) or an original, first and joint inventor (if plural names are listed below) of the subject matter which is claimed and for which a patent is sought on the invention entitled:

Method of Treatment for Central Nervous System Injury

(*Title of the Invention*)

the specification of which

☒ is attached hereto

OR

☒ was filed on (12/30/2003) [ ] as United States Application Number or PCT International

Application Number 10/748,572 and was amended on (MM/DD/YYYY) [ ] (if applicable)

I hereby state that I have reviewed and understand the contents of the above identified specification, including the claims, as amended by any amendment specifically referred to above.

I acknowledge the duty to disclose information which is material to patentability as defined in 37 CFR 1.56, including for continuation-in-part applications, material information which became available between the filing date of the prior application and the national or PCT international filing date of the continuation-in-part application.

I hereby appoint the practitioners at Customer Number 22446, who are the attorney(s) or agent(s) of the assignee of my invention to prosecute the above-identified application, and to transact all business in the United States Patent and Trademark Office connected therewith.

I hereby claim foreign priority benefits under 35 U.S.C. 119(a)-(d) or (f), or 365(b) of any foreign application(s) for patent, inventor's or plant breeder's rights certificate(s), or 365(a) of any PCT international application which designated at least one country other than the United States of America, listed below and have also identified below, by checking the box, any foreign application for patent, inventor's or plant breeder's rights certificate(s), or any PCT international application having a filing date before that of the application on which priority is claimed.

Prior Foreign Application Number(s)	Country	Foreign Filing Date (MM/DD/YYYY)	Priority Not Claimed	Certified Copy Attached? YES	NO
			☐	☐	☐

☒ Additional foreign application numbers are listed on a supplemental priority data sheet PTO/SB/02B attached hereto:

PTO/SB/01 (03-01)
Approved for use through 10/31/2002. OMB 0651-0032
U.S. Patent and Trademark Office; U.S. DEPARTMENT OF COMMERCE

DECLARATION AND POWER OF ATTORNEY Utility or Design Patent Application

Direct all correspondence to: ☐ Customer Number or Bar Code Label	22446	OR ☒ Correspondence address below	
Name Jill T. Powlick			
Address ICE MILLER, One American Square, Box 82001			
City Indianapolis	State IN	ZIP 46282-0200	
Country USA	Telephone (317) 236-5972	Fax (317) 236-2219	

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under 18 U.S.C. 1001 and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

NAME OF SOLE OR FIRST INVENTOR: ☐ A petition has been filed for this unsigned inventor			
Given Name (first and middle [if any]) Richard B.		Family Name or Surname Borgens	
Inventor's Signature			Date
Residence: City Delphi	State IN	Country US	Citizenship US
Mailing Address 1953 S. 900 W.			
City Delphi	State IN	ZIP 46923	Country US
NAME OF SECOND INVENTOR: ☐ A petition has been filed for this unsigned inventor			
Given Name (first and middle [if any]) Scott A.		Family Name or Surname Shapiro	
Inventor's Signature			Date 6/28/05
Residence: City Indianapolis	State IN	Country US	Citizenship US
Mailing Address 8826 Kirkham Road			
City Indianapolis	State IN	ZIP 46260	Country US

☐ Additional inventors are being named on the ___ supplemental Additional Inventor(s) sheet(s) PTO/SB/02A attached hereto.

853835.1

PTO/SB/01 (03-01)
Approved for use through 10/31/2002. OMB 0651-0032
U.S. Patent and Trademark Office; U.S. DEPARTMENT OF COMMERCE
Under the Paperwork Reduction Act of 1995, no persons are required to respond to a collection of information unless it contains a valid OMB control number.

DECLARATION AND POWER OF ATTORNEY **Utility or Design Patent Application**

Direct all correspondence to: ☐	Customer Number or Bar Code Label: 22446	OR ☒ Correspondence address below	
Name Jill T. Powlick			
Address ICE MILLER, One American Square, Box 82001			
City Indianapolis		State IN	ZIP 46282-0200
Country USA	Telephone (317) 236-5972		Fax (317) 236-2219

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under 18 U.S.C. 1001 and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

NAME OF SOLE OR FIRST INVENTOR:	☐ A petition has been filed for this unsigned inventor		
Given Name (first and middle [if any]) Richard B.		Family Name or Surname Borgens	
Inventor's Signature [signature]			Date 6/29/05
Residence: City Delphi	State IN	Country US	Citizenship US
Mailing Address 1953 S. 900 W.			
City Delphi	State IN	ZIP 46923	Country US
NAME OF SECOND INVENTOR:	☐ A petition has been filed for this unsigned inventor		
Given Name (first and middle [if any]) Scott A.		Family Name or Surname Shapiro	
Inventor's Signature			Date
Residence: City Indianapolis	State IN	Country US	Citizenship US
Mailing Address 8826 Kirkham Road			
City Indianapolis	State IN	ZIP 46260	Country US

☐ Additional inventors are being named on the ___ supplemental Additional Inventor(s) sheet(s) PTO/SB/02A attached hereto.

853835.1

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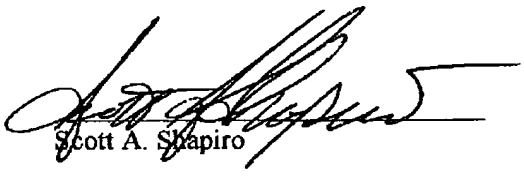
IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

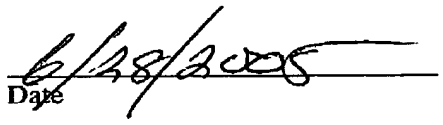
Re:	Invention:	METHOD OF TREATMENT FOR CENTRAL NERVOUS SYSTEM INJURY
	Inventors:	BORGENS, Richard B. and SHAPIRO, Scott A.
	Filed:	December 30, 2003
	Serial No.:	10/748,752
	Our File No.:	P01254-US-1

STATEMENT UNDER 37 C.F.R. § 1.48(a)(2)

I, the undersigned, hereby declare that the omission of my name as an inventor on the above-referenced patent application as originally filed occurred without deceptive intent on my part. I am signing, along with this document, a declaration for the above-referenced patent application. It is my understanding that each of the inventors listed above are the correct inventors for the above-referenced patent application.



Scott A. Shapiro



Date

INDY 1570498v1

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Re:	Application of:	BORGENS, Richard B., et al.
	Serial No.:	10/748,572
	Filed:	December 30, 2003
	For:	METHOD OF TREATMENT FOR CENTRAL NERVOUS SYSTEM INJURY
	Our File No.:	P01254-US-01 (19232.0011)

ASSENT OF ASSIGNEE TO CORRECTION AND/OR ADDITION TO ORIGINALLY NAMED INVENTORS

1. An Assignment of Invention for the above-referenced patent application for Richard B. Borgens, the named inventor, was recorded on June 21, 2004, Reel 014758, Frame 0770.

2. The Assignee, Purdue Research Foundation (an Indiana corporation), 3000 Kent Avenue, West Lafayette, Indiana 47906, assents to the correction of inventorship filed herewith.

3. Assignee Certification

In accordance with 37 C.F.R. § 3.73, the Assignee hereby certifies that the evidentiary documents with respect to its ownership have been reviewed and that, to the best of Assignee's knowledge and belief, title is in the Assignee seeking to take this action.

As a person signing below, I hereby declare that I am authorized to sign on behalf of the Assignee; that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

Purdue Research Foundation

By: Bruce L. Pershing Date: August 24, 2005

Name: Bruce L. Pershing

Title: Investment Officer and Corporate Secretary

INDY 1594751v.1

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